

SELECTION OF SUITABLE CASES.

It has been usual to regard with much suspicion any cases presenting evidence of infiltration or thickening in the lateral fornices or Douglas's pouch. But cancer does not only attack the normal pelvis, and a large number of women with old inflammation in the parametric tissues, pelvic peritoneum and Fallopian tubes, develop cancer of the cervix. Unless this is borne in mind a certain number of cases will be rejected which on abdominal exploration would have proved operable. Therefore, in a doubtful case it is good practice to examine, through an abdominal incision, the exact condition of the pelvis by touch and sight.

The question of infiltration of the bladder is far more important as regards the immediate and ultimate prognosis. In any doubtful case a definite opinion as to the advisability of proceeding with hysterectomy can only be given by opening the abdomen and seeing at once how far it is possible or advisable to strip the bladder from the cervix. Messrs. Berkeley and Bonney have pointed out that a wound of the bladder is a serious complication, extremely likely to occur if that viscus is involved in the growth, and very possibly fatal from the prolongation of the operation involved (cf. Case 6). In Case 8, however, a portion of bladder wall was resected, the gap sutured, and covered by a peritoneal flap. The patient recovered. Case 17 shows that involvement of the ureter is not necessarily an indication against the operation. The ureter was brought out in the left iliac fossa, and no bad symptoms ensued.

Pre-existing renal disease is perhaps a more serious complication in this operation than in many other surgical procedures. The handling and extensive dissection of the ureters, combined with the subsequent catheterization so often required, must lead to grave risk of a rapidly-ascending renal infection.

TECHNIQUE.

In reviewing the table of cases, the very striking fact is apparent that out of the 13 cases which recovered 6 developed suppuration in the abdominal incision; 1 in addition had an abscess in the pelvic cellular tissue pointing at the vaginal vault, and another an abscess, probably extraperitoneal, pointing in the right iliac fossa. These cases strongly suggest a technique in which there is room for improvement. Complete resterilization of the surgeon's and assistants' hands should be carried out immediately before suturing the pelvic peritoneal floor and abdominal incision, and for these steps a different tray of instruments should be reserved. The raw edges of the abdominal incision should be protected throughout the operation, as Berkeley and Bonney suggest, by sterilized cloths. At that most difficult and dangerous period of the operation, when the vagina is being divided, too much care cannot be expended on packing off the tissues around so that no cancerous particles or septic material may reach them. At this point I believe that the surgeon's and assistants' hands cannot possibly be any longer sterile.

In the majority of these cases a gauze drain has been pushed down from above into the open end of the vagina, and the cut edges of the latter brought together by suture, but it would seem to be the best practice to use no suture at all; it is in the extensive raw surface left in the sub-peritoneal cellular tissue that infection is liable to occur, and no drainage exit can be too large for this highly dangerous area; it is best ensured by the use of a large drainage tube which keeps the vaginal walls apart as long as may be necessary. A small gauze wick may be placed inside the tube, but if gauze alone be used the vaginal walls must tend to compress it and restrict the drainage. In those cases in which the cervix and vagina remain very dirty in spite of preliminary cauterization, etc., might it not be a certainty that infection of the neighbouring area takes place? Therefore drain freely.

Of the 13 cases which recovered, Nos. 3, 4, 5, 9, 14, 15, 16 could probably have been treated by vaginal hysterectomy; but the rest, either from the presence of infected glands or from adhesions and parametric infiltration, were too advanced for the vaginal route. Two cases have recurrence, one (No. 10) developing a mass in the left side of the pelvis about twelve months after operation, another having a mass in the vault of the vagina within a few months (No. 14). Of the 4 fatal cases, 3 were totally

unsuitable for vaginal hysterectomy, but one (No. 7) might have been so dealt with without loss of life. The abdominal route has therefore a far larger field in the removal or even in the palliation of the disease.

The operation itself would appear to be as complete as can be devised in this particular situation, but practically it is apt to fall short of the theoretical ideal which should govern all cancer operations. This ideal consists in the removal of the primary focus as widely as is consistent with safety together with the lymphatic and glandular area in one unbroken sheet. This ideal is well fulfilled in the modern operation for cancer of the breast—a situation in which it is anatomically possible to carry it out—but to remove iliac glands, parametrium, and uterus in continuity, and to avoid tearing across infected lymphatics is a matter of exceeding difficulty.

The commonly practised step of removing iliac glands after removal of the uterus implies that the lymphatic channels are torn across. The vagina should be divided at the lowest possible limit. It is surprising how the vaginal walls retract even when clamped, and how little of the vaginal wall appears to be removed when the specimen is examined after removal.

One more point is certainly fully deserving of recognition, and for the suggestion I am indebted to Mr. Charles Ryall. Before doing hysterectomy for cancer of the cervix, a sound should be passed into the uterus; this simple precaution may reveal the presence of a pyometra—a serious complication in the operation from the point of view of a sepsis.

REFERENCE.

¹ BRITISH MEDICAL JOURNAL, October 3rd, 1908.

ACCOUNT OF AN EPIDEMIC OF ENTERITIS CAUSED BY THE "LIVERPOOL VIRUS" RAT POISON.

BY

LIONEL HANDSON, and HERBERT WILLIAMS, M.B., B.S.Lond., M.D., D.P.H.,

MEDICAL OFFICER OF HEALTH, PORT OF LONDON.

WITH BACTERIOLOGICAL REPORT

By EDWARD KLEIN, M.D., F.R.S.,

LECTURER ON ADVANCED BACTERIOLOGY, ST. BARTHOLOMEW'S HOSPITAL MEDICAL SCHOOL.

THE following notes refer to an epidemic, confined to twelve persons, which recently occurred in a large business house in London. All the cases developed between July 18th and July 22nd inclusive. The actual incidence of the cases on each of these days was as follows:

	Cases.
On July 18th	1
On July 19th	7
On July 20th	1
On July 21st	2
On July 22nd	1

The disease was very severe amongst those who were first attacked—and, indeed, for the first day or two the condition of six of the earliest patients aroused the greatest anxiety, though eventually they all recovered. In the later cases the condition was progressively less severe, and in the patient who was last attacked the symptoms were quite mild.

SYMPTOMS.

The symptoms were the same in all cases, and only varied as regards severity. They were as follows:

Vertigo was the initial symptom complained of in every case.

Abdominal Cramps developed within an hour or two after the attack of giddiness, and in most of the cases the abdominal pain was extremely acute. When the first patients, some six in number, were originally seen, on July 19th, they were lying about on beds and sofas, with their knees drawn up, groaning, and obviously suffering very severely. These cramps, gradually becoming less acute, continued in the majority of cases for two or three days. In only one case did they persist for a longer

period. In many cases there was distinct tenderness on pressure over the left iliac region for several days.

Diarrhoea varied much in intensity. In the milder cases only two or three loose stools a day were passed, whilst in those who were more severely attacked, the diarrhoea was extremely frequent, and attended with so much tenesmus that the worst cases were seldom off the bed-slipper. The stools were very loose, watery, bile-stained, and contained small greenish pellets, about as large as a grain of wheat. In the severer cases considerable quantities of mucus were present, and in the worst cases of all there were streaks of bright blood.

Vomiting was very general, and in those more severely attacked continued for two or three days, occurring whenever any nourishment was taken.

The Tongue.—In all cases this was thickly coated with yellow fur, but in none did it become dry.

Thirst was universally complained of.

Urine was entirely suppressed in several of the cases for from twelve to twenty-four hours.

Collapse in the worse cases was a very pronounced feature. Their faces were pinched, ashen, covered with clammy sweat, whilst the pulse was small, and in many cases irregular.

Temperature.—In the more severe cases pyrexia was marked. In one case the thermometer registered 105°, in two others 104°, and in another 103°. In every case there was some elevation of the temperature, but in the milder ones it was not nearly so high. The temperature having quickly reached a maximum, fell steadily and continuously, so that in every case the normal was reached at the end of forty-eight hours.

Spleen.—In 3 cases only a slight enlargement of this organ was noticed.

Headache was greatly complained of during the first day or two.

The disease was of short duration. With the fall of the temperature the severity of the symptoms rapidly abated, and by the end of ten days all the patients were convalescent, though the majority of them had the appearance of having been through a very severe illness.

Specimens of the stools of some of the patients were sent to Professor Klein to be examined bacteriologically.

ETIOLOGY.

At the onset the cause of the epidemic was very obscure; and, indeed, it was not until the outbreak was over that the source of the infection was finally determined.

The very rapid succession in which the cases followed one another, and the fact that all those attacked lived in the same establishment and fed on the same food, seemed to point to the food as being the contaminating source.

The severity of the fever precluded ptomaine or other chemical poisons, and indicated that the infection was due to a living germ.

With a view to attempting to find out what particular article of diet was at fault, a searching inquiry was made as to the food that each of the patients had partaken of during the week preceding the epidemic. It is a rule of the establishment that the housekeeper must enter daily into a book, kept specially for the purpose, the *menu* supplied to each of the dining rooms. With the assistance of this book it was an easy matter for the patients to recall everything they had eaten. But no light was thrown on the subject, for all of them had eaten the same food, and every item of the *menu* had been eaten by each individual, and so might have been the infecting source.

Certain of the patients had also during the period had some meals outside the establishment. But the infection could not have been incurred in this way, for no two of the patients had had these supplementary meals at the same place, and three of the patients had had no food at all outside the establishment during the time under consideration.

No complaints were raised as to the appearance, taste, or smell of the food provided. Amongst the patients themselves some veal and ham pie provided for breakfast on July 17th was regarded with suspicion. But these suspicions were based solely on the fact that they had heard of veal and ham pie causing diarrhoea and vomiting on other occasions. As a matter of fact they all agreed

that this particular pie tasted excellent. Moreover, besides the twelve patients a very large number of other people lived on the premises, and none of them was taken ill, though pies identically prepared, and, indeed, all the other articles of food, had been eaten by them.

In the establishment there are five distinct dining-rooms in which the various divisions of the staff have their meals. On inquiry it was found that all those taken ill had their meals in the same dining-room, and that no illness arose amongst those using the other four dining-rooms. From this fact the conclusion was drawn that if, as appeared probable, the food was to blame, it must have become infected in the dining-room itself. For in all the other dining-rooms the diet was the same, and the food was all stored in central pantries, and cooked in and served from a central kitchen. No article of diet was served exclusively in the particular dining-room.

A careful inquiry was made amongst the kitchen staff and waiters to find out whether any of them had been ill recently, especially whether any of them had been suffering from diarrhoea, as it was thought possible that the food might have become infected through the hands of any one so affected if there had been any lack of personal cleanliness. But the kitchen staff and waiters had all been perfectly well.

At this time of the year flies were numerous, and the question of flyborne infection from outside was entertained. But nothing pointing to this as the source of infection could be discovered.

One fact seemed to point to a breakfast or supper as being the meal when the infection was contracted. All those who became ill were residents in the establishment, and had all their meals in. Besides these residents there was a large number who had dinner and tea only in the same dining-room, and as none of these was taken ill, the infection appeared to have occurred at some meal when they were not present.

Up to July 30th our investigations had led us to the following conclusions:

- (a) That the infection was due to a living germ.
- (b) That the infection was probably conveyed in the food.
- (c) That the infection of the food had probably taken place in the dining-room.
- (d) That probably the food at fault was eaten at breakfast or supper.

In the meantime Dr. Klein reported that his investigations had resulted in his being able to isolate the microbe causing infection, and that this microbe was neither *B. coli communis* nor Gaertner's bacillus.

We wish to emphasize the fact that up to July 30th we had no reason to connect the epidemic with the "Liverpool virus," for we were unaware that any rat poison had been used. Indeed, at this time we began to doubt whether we should succeed in clearing up the mystery, as with the exception of the above-mentioned conclusions we had been unable to find anything to further us in our inquiries.

On July 30th, however, a bad smell was noticed in a room quite close to the suspected dining-room. The flooring was taken up, and forty dead and decomposing mice were found. They were all lying alongside a hot-water pipe, where they had probably gone to get warmth on feeling ill.

We were then informed that mice had frequently been seen in the dining-room where the poisoning was suspected to have originated, and also in one of the pantries. On July 16th, two days before the first case developed, some "Liverpool virus" rat poison had been put down in these two places. The poison was only placed about on this one occasion, only in these two places, and only one tube of the virus was used. This virus consisted of a culture of an organism in a glass tube, and the method of using it was to spread some of the culture on bread, and to lay the bread about on the floors of the two rooms, where the mice could get at it. The food stored in this pantry, chiefly bread, butter, and cheese, was distributed indiscriminately amongst all the dining-rooms, and so it does not appear that it was in the pantry that the food was infected, but rather in the dining-room. The exact method by which the virus was transferred to the food is uncertain. Possibly flies may have been the medium. Possibly the mice may have got some of it on their feet, and then have run over the table-cloths, plates, etc.

Possibly the table-cloths, etc., may have been infected through the evacuations of the mice. Or possibly some milk, water, or other fluid may have been contaminated in one of the above ways.

But whatever the exact method by which the food became infected, the connexion between the "Liverpool virus" and the epidemic seems quite clear. For to recapitulate:

1. On July 16th "Liverpool virus" was put down.
 2. On July 18th, the epidemic broke out amongst those persons taking their meals in the room where the virus was placed.
 3. On July 30th, the virulence of the virus to the mice was proved by the discovery of the dead mice.
 4. Dr. Klein's investigations, as shown later on in the report, establish the identity of the microbe causing the disease with the microbe of "Liverpool virus."
- For the "Liverpool virus," which is stated to be a culture of a living bacillus, it is claimed:

1. That it produces a fatal contagious disease amongst rodents.
2. That the disease so produced compels the rodents affected to flee from houses in order to die in the open.
3. That it is harmless to domestic animals and human beings.

The facts observed by us abundantly establish the first claim, conclusively negative the second, and throw great doubt as to the correctness of the statement that the "virus" is harmless to human beings.

If our conclusions are correct, and it is difficult to see what other interpretation can be placed upon the facts—the use of the "virus" has in this instance been attended with very great danger to a large number of human beings. Luckily the persons affected were all young men, well nourished, well housed, and in robust health. Every care was taken of them during the time they were ill, for their employers spared no expense that they might be well nursed and receive every attention. Even under such favourable circumstances the gravest anxiety was caused in several cases, though, happily, they all recovered eventually. In less fortunate circumstances a fatal issue might easily have resulted.

It has appeared to us to be our duty to record the facts of this epidemic in detail in order to draw attention to the grave dangers that may attend the use of a culture of a living bacillus for the destruction of vermin. The sale and use of such poisons should be rigorously regulated.

Dr. Lauriston Shaw, who saw the majority of the patients in consultation at the beginning of the epidemic, has co-operated with us in describing the clinical features. He has since been made acquainted with the facts which later came to light, and he entirely concurs in the conclusions at which we have arrived.

DR. KLEIN'S REPORT.

It will be in your recollection that your Inspector brought here three stools from patients on July 22nd.

They had been voided and placed in sterile test tubes on July 21st. One further stool was brought here on July 23rd, voided on same day.

All four stools were fluid, and were labelled S. (1), L. (2), W. (3), S. (4).

Nos. 3 and 4 had a dark green appearance, No. 1 was quite colourless, and No. 2 had flakes and some blood pigment.

These stools were all dealt with in the same manner—namely, to discover in them the presence of any microbe of the Gaertner group generally associated with gastro-enteritic trouble, or of the microbe which predominated.

Of each stool five drops were diluted with 5 c.cm. of sterile salt solution, and then from this dilution one large and two small plates containing Drigalski-Conradi medium were made and incubated.

The result of the incubation of these plates was, briefly stated, thus: Besides some colonies of streptococci, the great majority, in fact the predominating number of colonies, were those of a microbe which was neither *B. coli communis* nor the Gaertner bacillus. Although it presented certain characters by which it might be considered either the one or the other, it possessed other characters by which it could be distinguished from both.

The accompanying table shows the cultural characters of the microbe, named here Microbe S. (1), and it will be noticed that it comes nearer to the Gaertner than to *B. coli communis*. In respect of the appearance of the colonies on Drigalski-Conradi medium, in surface and shake gelatine, in neutral-red broth, in litmus lactose peptone, in bile salt neutral-red agar, and also in respect of indol reaction, it materially differs from *B. coli communis*, whereas in respect of neutral-red broth, gelatine shake culture, and particularly in respect of agglutination, it differs from the Gaertner bacillus.

But the two points in which this Microbe S. (1) stands out as quite distinct and specifically important are the following:

1. Dr. Handson on July 25th was kind enough to supply me with three samples drawn in my presence with capillary tubes from three convalescents that had walked up to his office; a fourth sample from a convalescent was drawn and sent on July 31st. Neither of these convalescents was the patient S. from which Microbe S. was derived. A fifth sample from a different convalescent who had quite recovered was obtained on August 15th. This blood, however, when tested, had less pronounced, although distinct, agglutinating action than the blood of the convalescents of July 25th and July 31st.

The blood serum of these samples was tested for agglutination in dilutions of 1 in 20, 1 in 40, 1 in 60, and 1 in 100 on broth cultures (twenty-four hours incubated) on the following microbes: (1) *B. coli communis* of the same plates; (2) *B. enteritidis* (Gaertner) kept in subcultures in the laboratory; (3) the Microbe S. (1).

Nature of Test.	<i>B. Coli Communis</i> .	Gaertner's Bacillus.	Microbe S. (1).	Liverpool Virus.
Morphological	Short rods, fairly motile	Oval to cylindrical, very motile	Oval to cylindrical, very motile	Oval to cylindrical, very motile.
Surface gelatine	Dry, opaque	Dry, translucent	Dry, translucent	Dry, translucent.
Shake ordinary gelatine	Copious gas	Gas	No gas	No gas.
Shake glucose gelatine	Copious gas	Copious gas	Gas slight after three days	Gas very slight after three days.
Ordinary broth and phenol broth	Uniformly turbid	Uniformly turbid	Uniformly turbid	Uniformly turbid.
Indol reaction after four days	Positive	Positive	Negative	Negative.
Neutral red broth	Fluorescent, green	Fluorescent, green	Orange	Orange.
Litmus lactose peptone	Acid, gas	Negative	Negative	Negative.
Litmus milk (three days)	Acid, clotted	Alkaline, fluid	Alkaline, fluid	Alkaline, fluid.
Litmus lactose bile-salt peptone	Acid, gas	Bleached, no gas	Bleached, no gas	Bleached, no gas.
Litmus glucose bile-salt peptone	Acid, gas	Acid, gas	Acid, slight gas	Acid, slight gas.
Drigalski-Conradi plates	Red colonies with red halo	Blue colonies	Blue colonies	Blue colonies.
Bile salt neutral-red agar... ..	Red colonies	Negative	Negative	Negative.
Agglutination (broth culture twenty-four hours old) with convalescents' blood 1 in 60	Negative in one hour	Negative in one hour	Positive within twenty minutes	Partial, but distinct in thirty minutes.

The result of these tests was that the blood serum did not agglutinate either *B. coli communis* or *B. enteritidis* (Gaertner), but it markedly did so Microbe S. (1), this latter showing definite and distinct clumping within fifteen to twenty minutes; blood serum 1 in 60 and 1 in 100 agglutinated it completely within twenty minutes.

2. The second important point is this: A small guinea-pig was injected subcutaneously with about $\frac{1}{4}$ c.cm. of a forty-eight hours old broth culture of Microbe S. (1); next day it was quiet, showed a big swelling about the seat of inoculation, and did not take its food; the animal was found dead in about forty hours since injection.

On *post-mortem* examination it showed haemorrhagic oedema about the seat of inoculation—stomach, duodenum, and first part of jejunum pale and distended and filled with grumous fluid. This fluid on standing deposited flakes and turbid fluid; the flakes were composed entirely of detached masses of epithelium, so that we had here a condition like that found in cholera Asiatica.

From the blood of the heart and from the turbid fluid of the duodenum the microbe injected was isolated in great numbers. A blood culture was tested with blood of convalescent of July 31st, and showed distinct agglutination within thirty minutes in the same dilutions as above.

I received also on July 31st one agar culture of Liverpool virus.* This was used for studying and comparing it with the other microbes isolated from the stools and for agglutination test. As will be seen from the table, there is a distinct similarity in cultural respects between Microbe S. (1) and the Liverpool microbe. A difference between the two microbes—namely, S. (1) and Liverpool virus—appears in respect of agglutination, since the former agglutinated in a marked manner with convalescent's blood, whereas the latter showed under the same conditions a feeble reaction only. As a further important point it ought to be stated that the microbe, isolated in great numbers from the blood of the above injected guinea-pig, proved in cultural respects, as also in respect of agglutination, the same as the Microbe S. (1).

Subcutaneous injection of $\frac{1}{4}$ c.cm. of broth culture (twenty-four hours old) of the Liverpool virus into a large guinea-pig caused extensive gelatinous necrotic swelling in groin, abdomen, and chest, the animal after forty hours being quiet, off feed, and very ill. Exactly the same result was obtained in a companion large guinea-pig injected with $\frac{1}{4}$ c.cm. of broth culture of microbe S. (1).

E. KLEIN.

August 20th, 1903.

September 11th, 1903.

Since I sent in my report on the S. microbe and the Liverpool virus microbe I have made several series of comparative experiments which, I think, prove conclusively the identity of the two microbes.

The experiments are the following:

1. Both microbes injected subcutaneously separately into guinea-pigs cause the same kind of haemorrhagic oedema extending over large area.
2. Mice fed with either microbe (broth culture on bread) become ill and die in five to seven days, with the same *post-mortem* appearances.
3. Rabbits injected subcutaneously with either microbe developed extensive tumour; the animals are quiet and off food for several days, but are quite lively, and feed again by the end of the week.
4. Blood serum of a rabbit recovered, no matter whether S. microbe or Liverpool microbe has been used for injection, clumps in a marked manner a broth culture of twenty-four hours' incubation of both microbes almost instantly indiscriminately, that is to say, S. culture is clumped equally well by blood serum of S. rabbit or Liverpool rabbit, Liverpool microbe is clumped equally well by blood serum of Liverpool rabbit or S. rabbit.

E. KLEIN.

* The culture tube was sealed with paraffin, and, according to direction, was not to be used "later than August 17th, 1903."

A SPECIFIC SKIN ERUPTION IN PNEUMONIA.

By FRANK M. POPE, M.D., F.R.C.P.,

PHYSICIAN TO THE LEICESTER INFIRMARY.

WHETHER owing to improved diagnostic methods or because the pneumococcus is really changing its habits, we have become accustomed to thinking of it as an organism by no means confined to the pneumonic lung, but as able to grow well in joints, in the pericardial, pleural, and peritoneal cavities, in the meninges of the brain and spinal cord, and freely in the blood, producing a disease indistinguishable often from other forms of pyaemia. I do not remember hearing of its growth in the skin producing there a definite eruption, and therefore think the following case worthy of record:

H. S., a pale man aged 20, was admitted to the Leicester Infirmary on October 22nd. He had pneumonia and pleurisy of the base of the right lung. Temperature was about 104°. He had much delirium. Three days later he was worse. His heart was dilated, and a crop of papules was observed on the upper part of the back. They were of a deep rose colour, circular and about 3 to 4 mm. in diameter, darker than the average typhoid spot, and disappeared completely on pressure.

On October 28th the first spots were fading and a second crop identical with the first had appeared. The man was now very ill and had extensive consolidation of the base of the left lung.

On October 31st the spots were observed to be pustular. A smear of the pus was stained, and the only organisms to be seen were pneumococci.

A small abscess behind the right ear was opened on November 3rd. A culture was made from the pus; the growth consisted of pneumococci and staphylococci. A film taken directly from a pustule on the same day showed also pneumococci and staphylococci.

I can make but few remarks on the phenomenon. The eruption seems to resemble that of small-pox to some extent, as it was first papular and then pustular; a vesicular stage was not observed. Infection with staphylococci followed, as often in variola. The abscess appeared too late to be in causal connexion with the eruption. The patient made a slow but good recovery.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

PELVIC PATHOLOGY AND SEA-SICKNESS.

UNDER the influence of circumstances, certain pathological states are apt to be overlooked by medical men at sea when treating cases of sea-sickness.

It by no means follows that any case of vomiting occurring on the high seas is necessarily due to the fact that the sufferer happens to be on board ship. Especially is this so in the case of women subjects of a pelvic disorder to which they do not attach importance in connexion with their present ailment, and are naturally diffident about discussing it when being treated by the ship's surgeon for such a slight and common complaint as sea-sickness. The latter also, unless on his guard, is not likely to suspect extraneous causes and be content to run through the whole gamut of drugs and remedies in vain attempts to afford relief.

The following case is a typical example of this, and shows how a practitioner may be led astray, not so much by not looking as by not seeing:

Mrs. P., travelling to New Zealand, suffered continuously from sea-sickness, and none of the remedial measures usually employed seemed to have any effect. She had been rejecting everything taken into the stomach for over ten days—the weather during this time being sufficiently rough to account for it; there were other but milder cases on board—and the organ appeared to be in a state of acute irritation. There were no symptoms or signs pointing to a pathological condition, and beyond a "soreness," which could easily be attributed to constant vomiting, she complained of nothing. Nutrient enemata were resorted to in the hope of alleviating also the great thirst which existed, but even they set up a sort of reflex return.

On the twelfth day, while making her drink some thin water gruel so as to have something in her stomach to be "sick" with, I saw her actually vomit for the first time, and was immediately struck by its character—typically cerebral. Being quite certain that no cerebral mischief existed, she was asked, and consented, to have the pelvis examined bimanually, when the following conditions were found: Extreme congestion of parts, a badly torn cervix, an ovarian cyst, about the size of a hen's egg, on the left side, and finally a retroverted uterus.